AN ANALYSIS OF

UPPER GASTRO INTESTINAL BLEEDING

Dissertation Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

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for the award of the degree of

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CERTIFICATE

This is to certify that the dissertation titled **"AN ANALYSIS OF UPPER GASTRO INTESTINAL BLEEDING"** of **Dr. S.K. SUBHA KANESH** in partial fulfilment of the requirements for **M.S. Branch – I (General Surgery)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in September 2006. The period of study was from January 2004 to January 2006.

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DECLARATION

I, **Dr. S.K. SUBHA KANESH** solemnly declare that dissertation titled, "**AN ANALYSIS OF UPPER GASTRO INTESTINAL BLEEDING"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2004-2006 under the guidance and supervision of my Unit Chief

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The dissertation is submitted to Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.S. Degree (Branch – I) in General Surgery.**

Place : Chennai.

Date :

(Dr. S.K. SUBHA KANESH)

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PROFORMA

Patient Name : Age / Sex :

IP No / Ward :

Admitted with

1. Hemetemesis - Bouts
   - Coffee ground / Bright Red Vomiting
   - Quantity
   - Frequency
   - Duration since episode

2. H/o Malena / Bleeding PR / Diarrhoea / Constipation

3. H/o Drug intake (Analgesics / Steroids)

4. H/o Alcoholism / Smoking

5. H/o Jaundice / Ascites

6. H/o Epigastric Pain

7. H/o Previous bleeding episodes

8. H/o Known disease DM/HT/Bleeding diathesis

9. H/o Previous treatment

10. H/o Previous transfusion

11. H/o Metabolic derangements

O/E

Pulse rate

Blood Pressure

Respiratory rate

Temperature

Pallor

Jaundice

Hydration

Pedal edema

P/A Inspection

- Dilated veins
- Distension
- Shape
- Scar

Palpation

- Epigastric tenderness
- Hepatomegaly
- Splenomegaly
- Ascites
- Any mass
- Auscultation
- Bowel sounds

Per rectal examination

Investigation

1. Urine
    - Albumin
    - Sugar
    - Deposits
2. Blood
    - Hemoglobin
    - Grouping and typing
    - Bleeding time
    - Clotting time
    - Prothrombin time
    - TC, DC, ESR.

3.	Liver function tests	S. Bilirubin	-	Total
				Con jugated
				Unconjugated
		S. Proteins	-	Total
				Albumin
				Globulin
		S. Enzymes	-	SGOT, SGPT,
				SAP
4.	OGD Scopy	Oesophagus varices – Grade – Columns		
		Oesophagitis		
		Mallory weiss tear		
	Stomach	Diffuse erosion		
		Ulcer		
		Growth		

Treatment

- Conservative
  - Blood transfusion
  - Vasopressin
  - Sengs taken
  - Somatostatin
  - Saline lavage
- EST
- Surgery

Risk factor for rebleeding

Risk factors for morbidity and mortality

CONTENTS

INTRODUCTION

This dissertation is a randomized study conducted among patients with upper GI bleeding predominantly belonging to low socio economic status in Stanley Medical College during period January 2004 to January 2006.

UGI bleeding is one of the common and serious clinical problem related to severity of bleeding and its etiology.

The diagnosis and management of this emergency, role of general surgeon, remarkable value of endoscopy and other investigations are highlighted in this study.

The sex ratio, etiologic factors, key factors in management also high lighted.

This is one of the common surgical emergency with extremely frightening symptoms to the patients and relatives.

Alertness of surgeon with thorough understanding of causes of pathological and physiological factors, that determine the course, which will decide the outcome.

The ageing population, chronicity of disease like asthma, low backache, osteo arthritis, migrain, intake of aspirin, cortisone, NSAIDS, purchase of drugs across counter and using for longer duration are the accountable factors .

Its our aim to study incidence of etiology, clinical manifestation, course of illness, management of cases admitted to our hospital.

AIM OF THE STUDY

This study of upper gastro intestinal bleeding has been undertaken with view of analyzing the

- Etiology
- Various clinical presentations
- Methods of investigations
- Study of upper GI scopy
- Treatment in our hospital from January 2004 to January 2006.
- To determine age, dietary habits in distribution of disease categories.
- To analyse efficiency of investigations in localizing bleeding sites.
- To summarise treatment undertaken and to report their outcome.
- To study association of mortality with age, sex distribution of the disease.

THEORETICAL CONSIDERATION

Incidence varies from country to country. It is 1-2% of acute hospital admissions with an incidence of 170 per 100,000 adults per year.

The diversity of etiology with important causes being ulcer disease, erosive disease, esophageal varices due to portal hypertension.

CAUSES OF UPPER GI BLEEDING

Condition	%
ULCERS	60%
Esophageal	6%
Gastric	21%
Duodenal	33%
EROSION	26%
Oesophageal	13%
Gastric	9%
Duodenal	4%
OESOPHAGEAL VARICES	4%
MALLORY WEISS TEAR	4%
TUMORS	0.5%
VASCULAR LESIONS	0.5%
OTHERS	5%

Despite advanced sophisticated diagnostic and therapeutic techniques the overall mortality of UGI bleeding remain unchanged at 8-10%. The constancy of mortality rate probably reflects overall improved care of an aged, less fit patients population.

Virtually all studies indicate advanced age as predictor of mortality. Death among patients younger than 50 year becomes a rarity.

ANATOMIC CONSIDERATION

ANATOMY OF UPPER GI TRACT

Upper GI Tract consist of esophagus, stomach and duodenum.

OESOPHAGUS

Commences at lower edge of cricoid cartilage (C6 vertebra) and ends at oesophago gastric junction (T 11 vertebra).

This is a muscular tube, 25cm long, occupying the posterior mediastinum, 2cm lies below diaphragm.

It is closed at upper end by cricopharyngeus muscle 18 cm from the incisor teeth and at lower end by lower esophageal sphincter approximately 40 cm from incisor teeth.

The musculature of

- Upper 5% - including upper esophageal sphincter is striated.
- Middle 40% - mixed striated and smooth muscle.
- Distal 55% - is entirely smooth muscular.

LAND MARKS DURING ENDOSCOPY

- Cricopharyngeal sphincter which opens and closes intermittently.
- Left atrial distension may compress esophagus and displace it posteriorly.
- Left main bronchus – a tumor may cause narrowing of esophagus.

NERVE SUPPLY OF OESOPHAGUS

Vagus is the parasympathetic nerve supply which has synaptic connections to myentric (Auerbach's plexus).

In the body – there are only cholinergic receptors and vagal stimulation results in contractions.

In inferior oesophageal sphincter there are both cholinergic and adrenergic receptors, vagal stimulation results in relaxation of sphincter.

Afferent visceral pain impulses pass along sympathetic nerves which are closely related to somatic sensory fibres of phrenic and intercostal nerves in posterior horn of spinal cord.

ARTERIAL SUPPLY

Upper part	–	Inferior thyroid artery.
In its main extent	–	Oesophageal branches of aorta.
Lower part	–	Gastric and inferior phrenic arteries.

VENOUS DRAINAGE

- Cervical esophagus drain into inferior thyroid veins and then the brachiocephalic veins.
- Thoracic esophagus drains on left side into brachiocephalic vein via left hemiazygos and on right side through azygos system to superior vena cava.
- Abdominal esophagus drain into coronary, splenic, retroperitoneal and inferior phrenic veins which connect with portal and caval system.

LYMPHATIC DRAINAGE

Is longitudinal rather than segmental.

Lymphatic vessels in submucosa may run for some distances up and down before penetrating the muscle layers to join lymphatics in adventitia which drain to adjacent lymph nodes.

I) Para Oesophageal Nodes

Situated on the wall of esophagus include the cervical, upper, middle and lower thoracic para-esophageal and para cardial nodes.

II) Peri-Oesophageal Nodes

Located on structures immediately adjacent to esophagus, they include the deep cervical, scalene, paratracheal, subcarinal, posterior mediastinal, diaphragmatic, left gastric, lesser curvature and coeliac nodes.

III) Lateral Esophageal Nodes

Found lateral to esophagus and receive lymph from para and peri esophageal nodes. Includes posterior triangle, hilar, suprapyloric, common hepatic and greater curvature lymph nodes.

STOMACH

Main function is to act as reservoir for ingested food. It also serves to break down food stuffs mechanically and commence the process of digestion.

It originates as a dilatation in tubular embryonic foregut during 5^{th} week of gestation. By 7^{th} week, it descends rotates and further dilates with a disproportionate elongation of greater curvature into its normal anatomic shape and position.

Stomach is divided into

The Lesser Curvature

Continuous with the right free border of oesophagus, it is the concave border.

The Greater Curvature

Starts at cardiac notch,forms the convex border.

The Fundus

Area above the horizontal line from the cardiac notch. So it lies superior to oesophago gastric junction.

The Incisura Angularis

This is the junction of vertical and horizontal parts of lesser curvature clearly seen at gastroscopy.

The Body

Portion of stomach lying between fundus and pyloric portion.

The Pyloric Portion

Approximately distal one fifth of stomach consist of pyloric antrum, canal and sphincter. The pyloro duodenal junction is identified by vein of mayo.

HISTOLOGY

Histologically three types of mucosa can be recognized.

The Cardia

A small area of stomach around oesophago gastric junction, contains mucus-secreting glands only.

Fundic Gland Area

Lies between the pyloric gland area and cardia.

Gastric pits are shallow and mucosa contains parietal (acid secreting) and chief (pepsin secreting) cells.

Pyloric Gland Area

Forms mucosa of the pylorus. This area has deep gastric pits and contains mucus-secreting cells.

BLOOD SUPPLY

Stomach is richly endowed with an arterial supply on both lesser and greater curves. Most of the supply is from the ***Coeliac Artery***.

On the lesser curve

The left gastric artery, a branch of celiac axis anastamosis with,

The right gastric artery arises from the common hepatic artery.

On the greater curver

The right gastroepiploic artery

Arises from gastroduodenal artery which arises from hepatic artery and pass behind duodenum. *It is often this artery that is eroded in a bleeding duodenal ulceration*.

The left gastroepiploic artery

Arises from splenic artery and contributes to arterial archade along the greater curvature.

Fundus of stomach is supplied by vasa brevia or short gastric arteries which arise from near the termination of the splenic artery.

VENOUS DRAINAGE

Veins are equivalent to arteries, those along lesser curve ending in the portal vein and those along greater curve joining splenic vein.

Of particular importance is the left gastric or coronary vein, which receives branches from esophagus. This becomes markedly dilated in portal HT, and must be divided specifically in operations for bleeding esophageal varices.

LYMPHATIC DRAINAGE

Lymph nodes concerned in drainage of stomach are.

Hepatic Group

- Lie in lesser omentum along bile ducts receive lymph from liver and gall bladder. An outlying member along cystic artery is cystic node.

The Subpyloric Nodes

- Lie in angle between the 1st and 2nd parts of duodenum on head of pancreas in relation to bifurcation of gastroduodenal artery. They receive lymph from right 2/3rd of greater curve through inferior gastric nodes.

Gastric Group

- Superior node – along left gastric artery and paracardial nodes around cardiac end.
- Inferior node – lie along greater curve between layers of greater omentum.

Pancreaticolienal Group

- Lie along splenic artery in relation to upper border of pancreas. Some occur in gastrosplenic ligament in relation to short gastric branches.

NERVE SUPPLY

Has intrinsic and extrinsic supply

Intrinsic Nerves

- Principally the myenteric plexus of Auerbach and submucosal plexus of Meissner.

Extrinsic Nerve

- Derived mainly from vagus
- Vagal plexus around esophagus condenses to form anterior and posterior vagus. These are both afferent (sensory) and efferent fibres. Efferent fibres are involved in receptive relaxation of stomach and stimulation of

gastric motility as well as well known secretory function. The sympathetic supply is derived mainly from the celiac ganglion.

DUODENUM

It is 25 cm. Forms a 'C' loop with head of pancreas.

The first 2-5 cm of first part is entirely covered by peritoneum duodenal ulcers usually occur in this part of duodenum.

Ulcers in posterior wall penetrates, massive bleeding may occur if it erodes gastroduodenal artery.

Ulcers in anterior wall may perforate and cause generalized peritonitis.

Duodenum is lined by mucus secreting columnar epithelium. In addition Brunner's gland lie beneath the mucosa and are similar to pyloric glands in pyloric part of stomach. Endocrine cells in duodenum produce cholecystokinin and secretin.

LIGAMENT OF TREITZ

This is a supportive ligament containing unstriped muscle which pass to it from the region of left crus of diaphragm and the tissue about the celiac plexus. This is suspensory ligament of duodenum.

DEFINITION AND INCIDENCE

Upper gastro intestinal bleeding is defined as bleeding from a source proximal to ligament of Treitz.

It is a common and potentially deadly condition accounting for 85% of hospital admission for gastro intestinal bleeding.

Despite the availability of effective antiulcer medications and improved understanding of pathogenesis of ulcer disease, gastroduodenal ulcer disease remains the most common cause, responsible for half of bleeding episodes.

CLINICAL PRESENTATIONS

Hematemesis and melena are the most frequent clinical finding. However, massive bleeding from an upper source may also cause hematochezia.

ETIOLOGY

Important causes of upper gastro intestinal bleeding

1) Peptic ulcer disease which includes

- Gastric ulcer
- Duodenal ulcer
- Ulcer in stoma/anastamotic site
- Reflux esophagitis
- Ulcer in Meckel's

2) Oesophago gastric varices due to portal HT

3) Mucosal Erosions

- Gastric
- Duodenal
- Esophageal

4) NSAID – Associated disorder

Rare causes

1) Vascular Malformations

2) Dieulafoy's lesion

3) Mallory weiss tear

4) Aorto enteric fistula

5) Tumors of GIT

6) Hemobilia / Hemosuccus pancreaticus / pancreaticpseudocyst / aneurysm

PATHOGENESIS

MUCOSAL EROSIONS

Erosion is a break in mucosa that does not penetrate the muscularis mucosa and into submucosa. Often occur in stomach, may involve lower esophagus and duodenum.

At endoscopy the lesions are generally multiple with white bases, commonly encircled by a halo of erythema. When erosions have recently bleed their bases may be black.

Hemorrhage refers to appearance of discrete petichiae or bright red streaks and patches not associated with breaks in mucosa. Also called submucosal or subepithelial haemorrhage. Other confusing terms are hemorrhagic erosions acute gastritis and hemorrhagic gastritis. Erosive gastritis may be seen in

1) Stress lesions in seriously ill patients
    a. Organ system failure
    b. Burns
    c. CNS trauma
2) Drugs – Aspirin & NSAID, Alcohol, Corrosive ingestion, etc.

3) Trauma and physical agents
    a. Mechanical – Ryle's Tube, Retching
    b. Foreign body ingestion,
    c. Endoscopic hemostasis
    d. Radiation
4) Vascular
    a. Ischemia (embolism, Vasculitis)
    b. Congestive gastropathy
5) Reflux Injury
    a. Duodenogastric reflux : Post gastrectomy
    b. Gastroesophageal reflux
6) Idiopathic
    a. Chronic erosive gastritis (varioliform gastritis)
    b. Sporadic, incidental

Major cause of erosions in stomach is due to disturbances of gastric mucosal barrier. NSAIDs and alcohol are common causes.

Pathogenesis related to a combination of both gastric acid and activated pepsin injuring gastric mucosa, exacerbated by mucosal ischemia secondary to hypoperfusion.

PEPTIC ULCER DISEASE

This term includes gastric, duodenal, oesophageal, stomal ulcers, anastamotic ulcer, ulcer in Meckel's due to ectopic gastric mucosa.

Gastric and duodenal ulcer are common causes of UGI bleed. Duodenal bleed is more common than gastric ulcer.

H.Pylori infection remains most common etiological agent, followed by ingestion of ulcerogenic drugs and then stress. Other uncommon specific forms of peptic ulcer.

1) Acid hyper secretion
    a. Gastrinoma - inherited MEN I , sporadic
    b. Increased mast cells / basophils
    c. Antral G cell hyperfunction / hyperplasia
2) Other infections
    a. Viral infection – HSV Type I, CMV.
3) Duodenal obstruction / Disruption
    a. Congenital bands / Annular pancreas
4) Vascular insufficiency
    a. Crack cocain associated perforation
5) Radiation induced
6) Chemotherapy induced (hepatic artery infusions)

Major complications of peptic ulcer disease are intractability, hemorrhage, perforation, penetration and obstruction.

Bleeding ulcer is caused by acid-peptic erosions into submucosal or extraluminal vessels.

In stomach, bleeding vessel is typically a small submucosal artery.

In duodenum, posterior ulcer bleeds because of erosion of ulcer into retro peritoneal, extraluminal vasculature supplying duodenum and pancreas.

PORTAL HYPERTENSION

Normal portal venous pressure is 7-8 mm HG. When exceeds 10mm it is portal hypertension. In clinical practice portal pressure is measured indirectly as hepatic venous pressure gradient which is wedged hepatic minus free hepatic vein pressure.

Presence of varices at endoscopy or contrast imaging studies indicates portal hypertension

Portal venous system is entirely devoid of valves. Numerous small tributaries connect portal and systemic venous systems and evolve into major collateral channel when portal HT supervenes. Formation of collaterals is triggered when portal pressure rises above 12 mm Hg.

Important porto systemic channels that gives rise to UGI bleed

Left Gastric /coronary vein which connects oesophago cardiac venous plexus with splenic or portal vein. This is responsible for **esophageal varices**.

Short gastric and left gastro epiploic veins which connect the esophageal and gastric plexus with splenic vein. Responsible for formation of **fundal varices**.

HYPER DYNAMIC CIRCULATION OF PORTAL HYPERTENSION

Cirrhosis and other causes of portal HT
↓
Increased portal systemic resistance
↓
Portal Hypertension
↓
Increased production of vasoactive substances
↓
Portosystemic collateral formation
↓
Peripheral vasodilatation and increased plasma volume
↓
Hyperdynamic circulation

OESOPHAGEAL VARICES

Rupture of esophageal varices is more common than gastric fundal varices. Elegant studies revealed the peculiar portosystemic connection which occurs in distal 2-5 cm of esophagus.

Four distinct layers of veins noted.

- Intra epithelial veins connected to deep intrinsic vein via superior venous plexus.
- Deep intrinsic vein which is located in submucosa connected to para esophageal vein via perforating veins.

Major bleeding mainly due to rupture of deep intrinsic vein.

RUPTURE OF ESOPHAGEAL VARICES

Erosive Theory

- Due to reflux esophagitis.
- This theory was not accepted as antiulcer drug does not reduce incidence of ruptures.

Transmural Tension

- The difference in pressure between the esophageal lumen and that of the varix.

- Tension was inversely related to variceal wall thickness, derived by Laplace law $T = TP_X r/w$

TP - Transluminal pressure

r - Radius of lumen

w - Wall thickness

MALLORY WEISS SYNDROME

Forceful vomiting with a contracted cricopharyngeus and marked diaphragmatic fixation may lead to rise in intra abdominal and intragastric pressure that causes esophageal rupture.

This is prone in alcoholics. Lesion is characterized by a tear in proximal gastric mucosa near esophago gastric junction.

DIEULAFOY'S LESIONS

- Rare cause of GI bleed.
- lesions are unusually large submucosal or mucosal vessels found in gastric mucosa, most commonly along lesser curvature in mid-stomach.

AORTOENTERIC FISTULA

Uncommon condition, in which an inflammatory tract develops between aorta and gastro intestinal tract.

Develop as primary process resulting from infections aortitis, or inflammatory aortic aneurysm or a secondary process following aortic replacement with synthetic graft.

HEMOBILIA

Rare cause that results from blunt or penetrating hepatic injury with fistula formation with in the liver between vascular structure and biliary ductal system . Bleeding is usually mild and accompanied by jaundice and right hypochondrial pain.

HEMOSUCCUS PANCREATICUS / PANCREATIC PSEUDOCYST AND PSEUDO ANEURYSMS

UGI bleed can occur from variety of mechanism that are consequent to chronic pancreatitis. The autodigestive process imparted by pancreatitis can lead to pseudoaneurysm formation of any arteries such as splenic artery, gastroduodenal artery, pancreaticoduodenal artery, left gastric artery. Rupture of an artery results in bleeding.

UNCOMMON CAUSES

1. Chronic renal failure
2. Typhoid fever
3. Hemophilia A & B
4. Thrombocytopenia
5. Collagen Vascular Disease
6. Anti coagulant therapy.

CLINICAL FEATURES AND DIAGNOSIS

Thin, dark, granular coffee ground vomiting is important diagnostic feature of UGI bleeding.

Melena, hematochezia, blood streaked stools and occult blood in stools are clinical features but they may be confused with lower GI bleeding.

CLINICAL SIGNS OF BLOOD LOSS

American college of Surgery Committee on Trauma 1997 estimated fluid and blood loss in shock.

	< 1000ml	**1000 – 1500**	**1500 – 2000**	**> 2000**
BP	Normal	> 90. Sys.	70 – 90 Sys	< 90
HR	Normal	100 – 120	> 120 bpm	> 140
RR	Normal	> 24	↑	↑ / Hyperventilation
CRT	Normal	> 3 SLC	Cool, Pale skin	Cold, Skin
Urine	> 30ml/hr	25 – 30 ml/hr	5 – 15 cc ml/hr	Minimal
Mental changes	No	Weakness, Anxiety	+	Lethargic

Sources

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Nasogastric Aspirate Colour	Stool Colour	Mortality Rate (%)
Clear	Brown or Red	6
Coffee ground	Red	19.1
	Brown / Black	8.2
Red Blood	Black Brown Red	

Comparison Of UGI & LGI Symptoms		
	UGI	**LGI**
Hemetemesis	Assured	Ruled out
Melena	Probable	Possible
Hematochezia	Unlikely	Highly probable
Blood streaked stools	Ruled out	Assured
Occult blood in motion	Possible	Possible

Once diagnosis of UGI bleed was made first priority is rapid assessment of hemodynamic status.

Early symptoms of shock appears with blood losses of 15 – 20% includes weakness, cold skin, diaphoresis and pallor. These symptoms are due to sympathetic stimulation resulting from peripheral vasoconstriction.

When volume loss of 20-40% occur symptoms such as tachycardia, hypotension, mental confusion and oliguria (<400 ml/day) occurs.

When volume loss more than 40% profound shock manifested by prostration, depressed level of consciousness, thready pulse and blood pressure not recordable. Myocardial infarction followed by arrhythmias will be sequelae of profound shock in patient with old age with coronary artery disease.

INITIAL EVALUATION AND TREATMENT OF PATIENTS WITH UPPER GI BLEEDING

Four Primary Goals

1. Comprehensive patient assessment attention to hemodynamic status and identification of significant medical conditions.
2. Appropriate resuscitation and monitoring.
3. Identification of source of bleeding.
4. Specific therapeutic interventions to stop or control bleeding.

Initial patient assessment.

History

- Characteristic of bleeding
- Onset and duration of bleeding
- Associated symptoms
- Use of medications
- Significant medical conditions particularly liver disease.

Characteristic of Bleeding

- Vomiting of fresh blood
- Hemetemesis – Vomiting of blood or bloody gastric contents.
- Melena – Passage of dark tarry stools.
- Hematochezia– Passage of bright red blood from rectum.

Associated Symptoms

- H/O orthostatic dizziness or syncope indicate rapid and profound blood loss.
- Antecedent dyspepsia – suggestive of peptic ulcer disease.
- Crampy abdominal pain – more consistent with upper GI bleeding.
- Vomiting – may suggest Mallory – weiss tear.

History of Medications

- Salicylates or NSAIDS - may impair platelet function and contribute to poor coagulation.
- Warfarin
- Low molecular weight Heparin.
- Corticosteroids

Past Medical History

- Previous episodes of GI bleeding.
- H/O dysphagia or reflux esophagitis.
- Recent GI distress with vomiting.
- Peptic ulceration / H.Pylori infection.
- Liver disease, alcohol abuse.

- IBD, intestinal polyps, malignancy.

Co-Morbid Medical Conditions

- Renal insufficiency
- Atherosclerotic disease
- Congestive cardiac failure
- Chronic respiratory conditions
- Preexisting liver disease
- CNS disability

Physical Examination

Major initial objective is to determine the degree of blood loss and volume depletion.

LABORATORY ASSESSMENT.

Blood Investigation

- Hemoglobin
- Hematocrit
- Coagulation Profile
- Blood grouping and typing
- Bleeding Time
- Clotting Time
- Prothrombin Time
- Platelet count

These help to plan for transfusion to rule out bleeding and collagen vascular disease.

Liver Function Test

- S. Bilirubin
- SGOT
- SGPT
- SAP
- GGT

Help for management

Gives clue to diagnosis and etiology

Abnormal LFT indicates bad prognosis

Child Pugh's Criteria

	5-6 (A)	**7-9 (B)**	**10 – 15 (C)**
S. Bilirubin (mg/dl)	<2	2-3	>3
S. Albumin (gm/dl)	> 3.5	2.8-3.5	>3
Prothrombin Time	1-4	4-6	>6
Ascites	Absent	Slight	Moderate
Encephalopalthy	None	Minimal to moderate	Coma

A - Good liver function

B - Moderate impairment

C - Poor liver function

A & B - 70 – 80% survival rate

C - 30% survival rate

- S. Electrolytes

- Renal function tests
- Upper GI scopy
- Angiography

UPPER GI ENDOSCOPY

Gold Standard in diagnosis of upper GI bleeding.

INDICATION FOR EMERGENCY ENDOSCOPY

Patients with active hematemesis and massive melena with hypotension and hemodynamic instability should undergo endoscopy as soon as possible after proper resuscitation.

Patient Preparation

- First establish a patent airway as massive bleeding may predispose to aspiration.
- Hemodynamic status assessed
- Gastric Lavage

Not necessary in a majority of patients because blood tends to pool in the fundus when patient is lying in left lateral position.

Since most causes of bleeding are located in lesser curve and duodenum, adequate visualization possible without disturbing the pool of blood.

If gastric lavage necessary a large bore sump tube with large side holes should be used rather than the small nasogastric tubes which can be blocked easily by clots.

Sedation for Endoscopy

- Sedation necessary to calm and relax the patient especially since therapeutic endoscopy is longer compared to diagnostic endoscopy.
- Commonly IV benzodiazepines are used.

Procedure

- Endoscopy is performed under IV sedation and with topical pharyngeal anaesthesia.
- Patient in the left lateral position.
- Adequate suctioning is important to remove oropharyngeal secretion and in case patient vomits.

Passing the endoscope

There are two methods 1) Blind method, 2) Direct vision method.

Blind Method

Tip of endoscopy passed into patients mouth through a bite guard and advancing the tip approximately 18-20 cm from incisor teeth. Patient is asked to swallow, cricopharyngeal sphincter opens tip is advanced into esophagus by bending the tip of endoscope.

Direct Vision Method :

Passing endoscope through a bite guard and watching endoscopically to observe anatomy of hypopharynx.

After passing through cricipharyngeus, important land marks are

- Oesophago gastric junction
- Cardia
- Incisura
- Pylorus
- Superior duodenal flexure

OESOPHAGUS (20-40cms from incisor)

Important landmark are

- Indentation of left main bronchus
- Pulsation of left atrium
- Pulsation of aorta

Mucosa

- Mucosal colour
- The folds which run parallel and converge to become cardiac rosette.

Oesophago Gastric Junction

Seen where pale pink mucosa meets the dark red gastric mucosa, often seen as irregular 'Z' line- ora serrata.

The diaphragm normally clasps oesophago gastric junction and position of diaphragm and hiatus should be noted. When patient is taking deep breath, varices are seen as red / blue raised columns in lower oesophagus.

STOMACH

With slight angulation to left and anteriorly endoscope is passed in to stomach. Fluid removed to improve visualization and to reduce aspiration. It is important to note presence of bile, food or blood and the following

- Ulcer
- Growth
- Fundal varices are diagnosed

Fundus

On retroflexion, fundus appear as hollow rounded area with flat mucosa. Mucosal capillaries are prominent while venules are smaller and straight. Gastric varices are wide and appear serpigenous.

Gastric Ulcer

With patient lying on his left side fluid will collect along most dependent portion of greater curve. This is called as gastric lake.

The fluid is usually grayish in the absence of bile but greenish yellow in presence of bile.

Body

Rugal folds principally located in greater curve. In lesser curve they are vertically placed.

Gastric Angle

- Major landmark for lesser curve and separation of body and antrum.
- Lesser curve ends distally as a cresent shaped mucosal fold called incisura.

Pylorus

- Starts from incisura
- Pyloric antrum is identified by horizontally placed rugal folds.
- Pyloric channel is within 2 cm of pylorus with enormous variable.

DUODENUM

- Duodenal bulb
- Triangular base is demonstrated by angular fornices which are best seen during retroflexion. .

Mucosa of Duodenum

Mucosa is smooth. Arranged in concentric fold or triangular fold called “valves of Kerking”.

In emergency after adequate suctioning

- Oesophageal pathologies such as varices, ulcers, tumours Mallory-weiss tears should be obvious.
- Fresh blood refluxing into esophagus indicate active bleeding lesion in stomach.
- With endoscope in stomach air is insufflated to distend stomach lumen and provide an adequate endoscopic view.
- In most cases it is useless to attempt removal of blood or clots in fundus, rather examine by sliding the endoscope along lesser curve above the pool of blood until it reaches II part of duodenum.
- Most bleeding pathologies readily seen since they lie along lesser curve in distal antrum and duodenum.
- If no pathology is found, return the endoscope into stomach aspirate the fluid portion of blood pooled in corpus and fundus. Turn the patient to right lateral position. This shifts the blood and clot in to distal stomach to allow better examination of fundus.
- Adherent clots removed to expose the underlying bleeding pathology.

PROGNOSTIC FINDINGS AT ENDOSCOPY FOR PEPTIC ULCER

Actual appearance of ulcer at endoscopy is the most important predictor of rebleeding.

APPEARANCES OF ULCERS

- A clean ulcer base
- A flat ulcer
- Pigmented spot (Purple / Browns / Black)
- Adherent clot
- A visible vessel which may be smooth surfaced or tubular protuberance on ulcer surface or active bleeding with sprouting blood, continuous oozing or oozing around an adherent clot.

Later four appearances are considered stigmata of hemorrhage

PROBALILITY OF REBLEEDING.

- Clear ulcer base – rarely bleeds.
- Flat / pigmented spot – 10%
- Adherent non bleeding clot – 20%
- Visible vessel – 40 – 80%
- Ulcer size >1 cm

STIGMATA OF RECENT BLEEDING

Forrest Classification System

Descriptive identification of ulcer characteristics

F Ia - Active spurting bleeding ulcer

F Ib - Non-spurting active bleeding ulcer

F II a - Ulcer with a visible vessel or pigmented protuberance.

F II b - Ulcer with adherent clot

F II c - Ulcer with pigmented spot

F III - Clean ulcer base without stigmata of bleeding

An adherent clot and a non-bleeding visible vessel are considered major stigmata and carry high incidence of rebleeding (30-40%).

Ulcer with clean base the risk of rebleeding is less than 5%.

FINDINGS AT ENDOSCOPY FOR ESOPHAGEAL VARICES

GRADING OF OESOPHAGEAL VARICES

Grade I - Varices can be depressed by endoscope

Grade II - Varices cannot be depressed by endoscope

Grade III - Varices are confluent around the circumference of the esophagus.

GRADING OF VARICES AT 2 CM ABOVE THE OESOPHAGO GASTRIC JUNCTION

Grade	**Bite width of 1.7mm, biopsy forceps 5mm**
I	$1/4^{th}$ bite width
II	½ bite width
III	$3/4^{th}$ bite width
IV	1 or more bite width

PREDICTORS OF BLEEDING VARICES

Size of varix

- Directly proportional to rupture of varix

Colour

- Red colour
- Important predictor

Cherry red spot

Dilated subepithelial veins may appear as raised cherry red spot. The hemocystic spot is 4 mm in diameter. It typically lies in summit of dilated veins. It represents blood coming from deeper extrinsic veins of esophagus straight out toward the lumen through communicating vein into superficial submucosal vein.

These 3 signs are highly positive predictors.

SIGNS OF RECENT BLEEDING

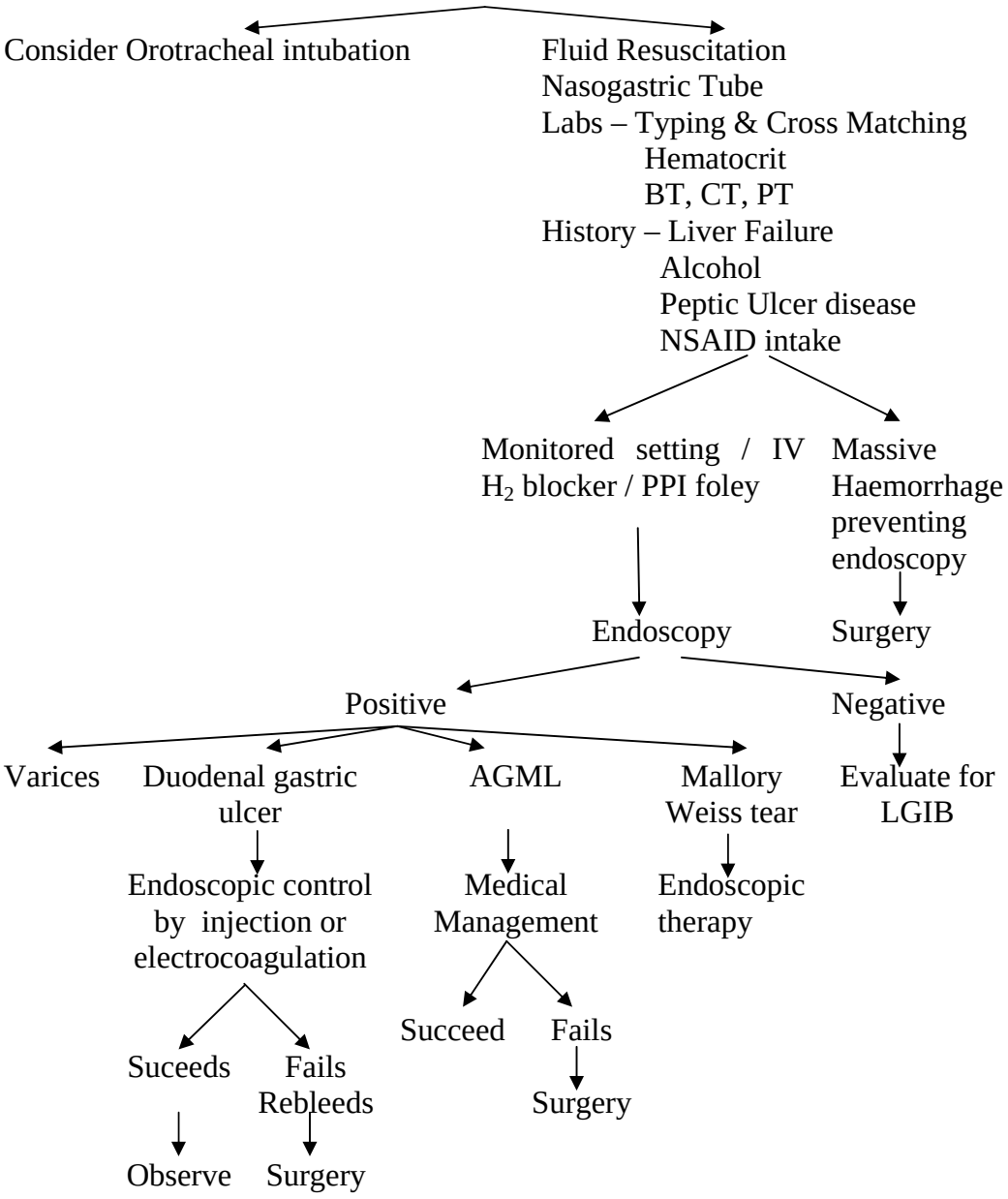
- Fresh clot in the base
- Pulsation of vessel

VARIOUS TECHNIQUES CURRENTLY AVAILABLE FOR ACHIEVING HAEMOSTASIS

- Injection of vasoactive agents.
- Injection of sclerosing agents
- Bipolar electrocoagulation
- Band ligation
- Thermal probe coagulation
- Constant probe press tamponade
- Argon plasma coagulator
- Laser photo coagulation
- Rubber band ligation
- Application of haemostatic material including biologic glue.

ALGORITHM FOR MANAGEMENT OF UPPER GI BLEED

ACUTE UPPER GASTRO INTESTINAL HAEMORRHAGE



AGML: Active Gastric Mucosal Lesion
LGIB : Lower gastrointestinal Bleeding

MANAGEMENT OF VARIOUS CAUSES

Mucosal Erosive Disease

Refers to endoscopically visualized lesions (subepithelial haemorrhage and erosions) that may cause bleeding. As these lesions are strictly mucosal lesions whereas all blood vessels are significant size or in submucosa and below they never have life threatening bleeding or large transfusion requirement

Main mode of management is conservative line of medical management. Endoscopic therapy is unlikely to be helpful or needed because of the diffuse, superficial nature.

Peptic Ulcer

Peptic ulcer and duodenal ulcers are most common cause of acute hemorrhage in upper gastrointestinal tract accounting for 25% of cases.

About 5% of patients with peptic ulcer disease have hemorrhage as the initial manifestation of the condition.

Hemorrhage remains most lethal form of complicated ulcer disease.

JOHNSON'S CLASSIFICATION OF PEPTIC ULCER

Type I

- Gastric Ulcer
- 60 – 70%
- Location - lesser curvature at or proximal to incisura, near junction of oxyntic and antral mucosa.
- Associated with diffuse antral gastritis or multifocal atrophic gastritis.

Type II

- Gastric ulcer associated with active or chronic duodenal ulcer
- Location – same site

Type III

- Pyloric Channel Ulcer
- Location with in 2 cm of pylorus

Type IV

- Gastric ulcer
- Located in proximal stomach or in the gastric cardia.

Type 2 and 3 gastric ulcers appear to behave more like duodenal ulcers and associated with excess acid.

Type I and IV are not associated with excess acid.

NSAID induced ulcer. They are typically multiple and located anywhere in stomach where protective mucosal barrier was damaged.

PORTAL HT GASTROPATHY

Seen largely in fundus, but can extend through out stomach. Shown as mosaic – like pattern with small polygonal areas surrounded by whitish yellow depressed border. Red point lesion and cherry red spots predict high risk of bleeding. Black brown spots are due to intra mucosal hemorrhage.

Sclerotherapy increases the gastropathy in portal hypertension.

Management

- Resuscitation
- IV cannulation – fluids
- Blood transfusion
- Oxygen mask / Nasal cannula
- Ryle's Tube
- Continuous Bladder Drainage
- Endoscopy
    - Clean based ulcer-discharged home after resuscitation, stabilization and institution of ulcer therapy.
    - Flat spots or adherent clots – watched in hospital , as recurrent bleeding is low

- Visible vessel and active bleeding- to undergo endoscopic hemostatic therapy.

Surgery for Gastric Ulcer

- Patients with severe ongoing ulcer bleeding despite attempt at endoscopic therapy.

Procedures used

- Ulcer excision with or without antrectomy
- Partial gastrectomy, with Billroth I anastomosis
- Partial gastrectomy with Billroth II anastomosis
- Cseudes procedure

Surgery for bleeding duodenal ulcer

- Suture ligation of bleeding artery with vagotomy with drainage procedure.

LONG TERM PREVENTION OF ULCER BLEEDING

- Conservative medical therapy
- Full course of antiulcer therapy

OESOPHAGO GASTRIC VARICES

Bleeding from osophagogastric varices responsible for one third of all deaths in patients with cirrhosis and portal hypertension.

90% of cirrhotic patients develop esophageal varices and 25-30% develop hemorrhage. Repeated hemorrhage develop in 70% of patients.

MANAGEMENT

Upper GI scopy remains main stay in investigation and treatment

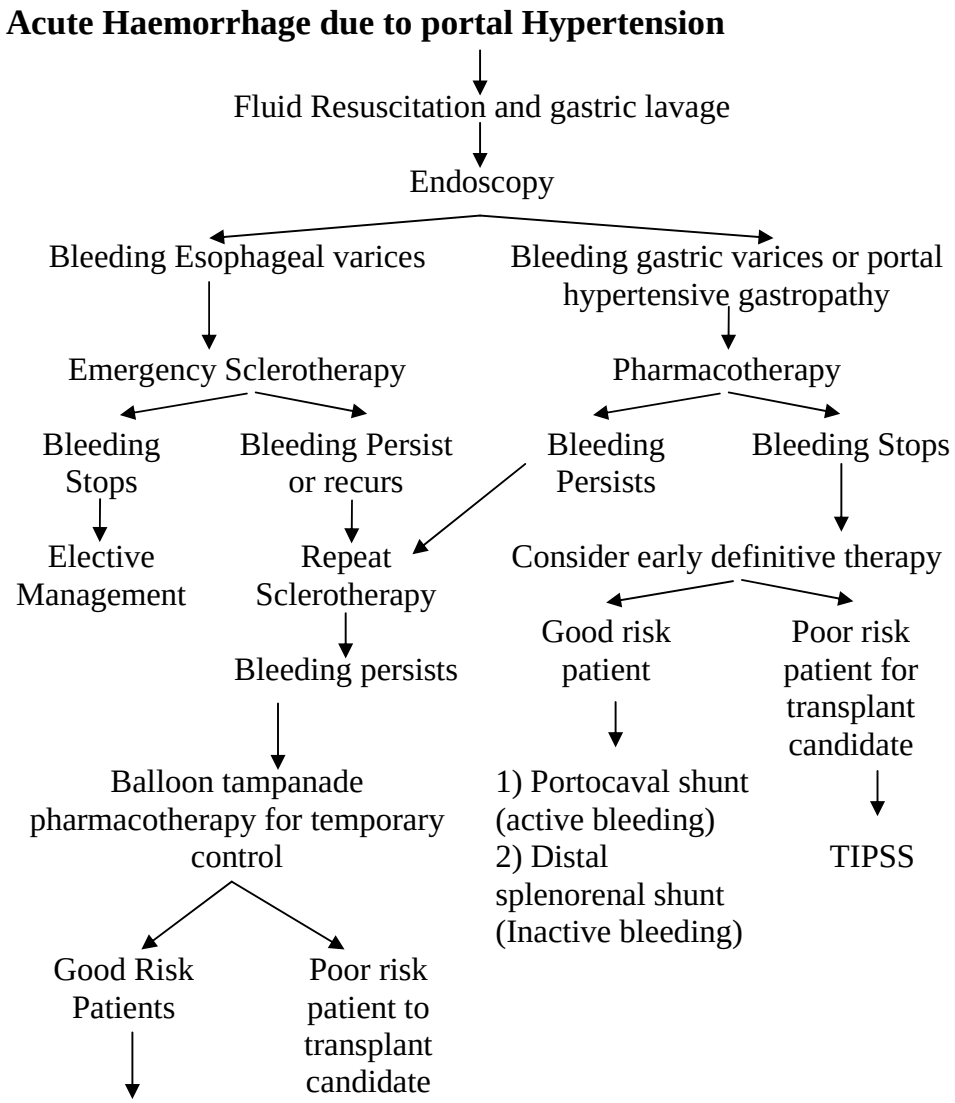
MEDICAL MANAGEMENT

- Vitamin k IM route
- Platelets, fresh frozen plasma infusion
- Octreotide infusion
- Sengstaken – Blackmore tube
- Minnesota tube
- Endoscopic – Sclerotherapy

  - Band ligation

ALGORITHM FOR MANAGEMENT OF ACUTE

HEMORRHAGE DUE TO PORTAL HYPERTENSION



1. Portocaval shunt (active bleeding,)
2. Distal splenorenal shunt (Inactive bleeding)
3. Esophageal transection.

↓

TIPSS

TIPSS : Trans jugular intrahepatic porto systemic shunt surgery

SURGICAL MANAGEMENT

1. Oesophago gastric devascularisation with or without splenectomy (Nonshunt)

2. Surgical decompression of portal venous system by shunt operation.

Indication : Two sessions of failed sclerotherapy with reasonable liver failure,

Porto systemic shunt surgeries

- End to side portocaval
- Proximal splenorenal shunt
- Distal splenorenal shunt (Warrant's shunt)
- Transjugular intrahepatic portacaval shunt (TIPS)

NON SHUNT SURGERIES

Splenic vein thrombosis with isolated fundal varices are best treated by splenectomy with or without devascularisation procedure.

OTHER OPERATIONS

- Oesophageal transection
- Porto Azygos disconnection by Hassab procedure – Devascularisation of upper half of stomach and esophagus.
- Sugiura's procedures

I Stage :

Devascularisation of upper half of stomach and esophagus as in Hassab procedure.

II Stage :

Vagotomy with pyloroplasty was performed.

CASUES OF MORBIDITY AND MORTALITY

Most important cause of death in upper GI bleed is shock and its sequelae. Comorbid medical diseases particularly myocardial ischaemia and infarction are aggrevated due to shock leading to fatal arrhythmias.

Mortality associated with underlying medical illness and gastro intestinal bleeding.

Condition	Mortality Rate (%)
Renal disease	29
Acute renal failure	63
Liver disease	25
Jaundice	42
Pulmonary disease	23
Respiratory failure	57
Cardiac disease	13
Congestive heart failure	28

Risk factors for morbidity and mortality

1. Age > 60 years
2. Shock at initial presentation
3. Transfusion requiring six or more units of blood
4. Coronary artery disease
5. Chronic pulmonary artery disease
6. Acute pulmonary failure

7. Acute and chronic renal failure
8. Cirrhosis
9. Acute hepatic failure
10. Sepsis
11. MODS
12. Coagulopathy
13. Recent CVA
14. Malignancy
15. Immunosuppression
16. Post operative stage

Prognostic indicators for mortality from peptic ulcer hemorrhage.

Clinical Parameter	Mortality Rate (%)
Overall	5-8
Age	10-15
≥ 60 years ≥ 80 years	25 – 30
Systolic blood pressure on presentation	
80 – 90 mm Hg	12 – 15
< 80 mm Hg	30 – 35
Nasogastric aspirate on presentation	
Coffee – ground appearance	6 – 10
Red Blood	18 – 20
Transfusion requirement	
≥ 10 units	28 - 34

REVIEW OF LITERATURE

1) Upper GI bleeding in a Brazilian Hospital, a retrospective study of endoscopic records by . Zaltman C, Souza Hs, Dept. of Int. Medicine, Federal University of Riode Janeiro, Brazil.

Aim : To assess clinical characteristics, endoscopic accuracy, treatment efficacy and clinical out come of patients.

Results :

- Most patients were male 68.7% mean age 54.$\pm$17.5.
- Bleeding site detected in 75.6% patients.
- Diagnostic accuracy greater within 24 hours of bleeding onset and in presence of hemetemesis.
- Peptic ulcer main cause of upper GI bleed (35%)
- Variceal bleeding (20.45%) indicating higher rate of underlying liver disease.
- Endoscopic treatment performed in 2

  3.86 % patients.
- Permanent hemostasis – 86% at 1st intervention 62.5% after rebleeding.
- Emergency surgery was seldom necessary.
- Average number of blood units 1.44 + 1.99 / patients.
- Average length of hospital stay 7.71 + 12.2

- Rebleeding in of in 9.1% patients.
- Mortality rate 15.34% significant with liver disease.

Conclusion

Diagnostic accuracy were related to time interval between bleeding episode and endoscopy and to clinical presentation. Endoscopic therapy an effective tool for selected patients.

Increased duration of hospital stay and higher mortality rate in patients subjected to therapeutic endoscopy attributed to higher prevalence of variceal bleed and liver disease.

2) Literature Survey of Causes of UGI Bleed

	Morjan et al % OMGE Study 1986	**Siverstein % ASGE study 1981**	**Phillip European study 1980**	**Kohler and Riemann 1989**	**Zaltman C et al**
DU	36	22.8	52.4	29	40
GU	-	21.9	-	24.0	7.5
Gastric Erosion	6.9	29.6	-	11.0	15.4
Esophogitis	4.1	12.8	-	5.0	7.6
Eso.Varices	13.4	15.4	11.2	14.0	5.6
Mallory Weiss	2.4	8.0	-	5.0	2.1
Neoplasm	2.6	3.7	9.8	4.0	2.3
Du Erosion	-	9.1	-	4.0	12.4
Stromal Ulcer	-	1.9	-	5.0	0.9
NSAID	-	42.2	321.4	15.6	28.6
Mortality	8.3	10.8	5.8	-	6.9

3) Etiology of UGI bleeding Jordanian patients, a prospective study.

Mustafa Mohennak MD, Department of Int. Medicine, GI & Liver Unit, Jordan, University Hospital & A1 Bashir Hospital, Amman.

Conclusion :

1. Emergency endoscopy should be performed in all.
2. High risk patients transfer to special centre
3. DU disease more common.

4) Retrospective review of emergency department patients with non variceal UGI bleeding for potential OP management.

Kum-ying Tham et al, Dept. of emergency medicine Tantock Seng Hosp. Singapore. Dept. of Emergency and Critical Care Medicine Keio University School of Medicine and Keio University Hospital, Tokyo, Japan. Dept. of Emergency Medicine, Massachusetts General Hospital, Boston, MA.

Objective : To determine number of ED patients with non variceal UGI bleeding who could have been managed as OP's through previously developed clinical guidelines.

Results :

145 patients seen 128 admitted 111 (77%) underwent OGD, 21 (91%) had varices, 90 (81%) with non variceal UGI bleeding 18 of these 90 fulfilled guidelines for OP management and none of 18 had complication.

Conclusion :

In non HMO urban teaching hospital 18 patients with non variceal UGI bleeding met criteria for OP management in 6 month

period and none developed complication during a mean in hospital – stay of 21 day.

5) Acute UGI bleeding in Kuwait – 1995; Kuwait Medical Journal. 2001, 33(2) (44-14) Abdul Karrem. Y. Khajah et al, Gastroentrology Department , Al-Amiri. Hospital – Retrospective study.

Results :

215 case admitted in 1995 overall incidence 28.3 per 100,000 .

Most common case – Peptic Ulcer Disease (19%) Esophageal Varices (7%) 10 fatalities, due to 10- morbidities.

Conclusion :

UGI bleeding in Kuwait is most commonly due to peptic ulcer disease and varices mortality rate lower than that from developed countries.

6) UGI Bleeding in Medical Intensive Care Unit, Doha, Qatar- Kamaha A et al, Dept. of Medicine, Medical Intensive Care Unit. Hamad Medical Corporation, Doha, Qatar. QMJ, June 2003.

UGI Bleed is a common problem, and important cause of morbidity and mortality study from June 1999 to May 2000, 860 patient admitted 102 with UGIB. Common age group – 50 – 60 years (28.4%) Frequent cause – Peptic ulcer disease 50 patients followed by variceal bleeding.

7) Upper GI bleeding in An Urban Teaching Hospital 1992 – 1993, 2002 – 2003, DG Morgan, J. Belback, M Lees. Mc Master University Division of GE, Hamilton, Ontario.

He studied for 10 years and results were n (223, 146)

Results	1992 – 1993	2002-2003
Age	60.5 years	70.5 years
Female	58%	45.9%
NSAID	41%	0.2%
PPI	12.5%	15.1%
H2RA	30.5%	5.8%
Mortality	1.8%	13.7%
Surgery	12%	5.8%
Hematamesis at presentation	58%	46%

Conclusion :

Few patients were diagnosed with upper GI bleed before. Population is older and higher proportion of male. NSAID use is higher now than decade ago. Population was older, more NSAID use, medical problem and do not have gastric prophylaxis could contribute to increase in mortality. More surgery some decade age. While there is reduction in total no of patients with UGI bleed between 2 groups these patients still remain a large burden for health care system

8) Cooper et al studied effectiveness of performing an early endoscopy with in first 24 hours of acute UGIB episode.

Early endoscopy associated with reduction in length of hospital stay, rate of recurrent bleeding and need for surgical intervention.

9) Upper GI bleed an Etiological study of 552 cases Journal Pakistan Institute of Medical Science, July 2004. 15 (1): 845 – 8. Study conducted between 1992 – 2000. in Dept. of GE at PIMS. Tashfeen et al.

Oesophageal varices accounted for majority of lesions causing upper GI bleeding (44%). Peptic ulcer disease second commonest (19.7%), Oesophageal lesions like oesophagitis and esophageal ulcers 6.6%, Tumors of UGI 1.1%, Gastric erosion 4.5. Age variation maximum no of patient 50 – 59 years (22.6%).

It was concluded that causes of UGIB are similar to those in local literature but different from causes described in Western literature.

10) Upper GI bleed in an urban hospital, etiology, recurrence and prognosis. Ann. of surgery 1990, Oct, 212(4) 521 – 6.

Sugawa C et al. Dept. of Surgery, Wayne State University, Detroit, M1 48201.

Acute UGIB common cause of hospital admission and morbidity and mortality.

This study reviewed 469 patients.

Most common cause of bleeding, include gastric mucosal lesion 135 patients (24%) esophageal varices (221). Gastric ulcer 108 patient s(19%). DU 78 (14%). Non operative treatment in 89.5%. Endoscopic treatment in 144 cases. Operations performed in 58 cases (10.5%). Emergency operation to control bleeding in 2.5% cases. There were 58 deaths to UGIB factors correlating with death include shock at admission transfusion requirement more than 5 units.

MATERIALS AND METHODS

Present study made on 100 cases of UGI bleed admitted in our Stanley Medical College from January 2004 to January 2006.

Proforma made and careful entry made for each case.

Analysis of case regarding age, sex, OGD finding and variety of treatment opted and conclusion drawn.

All patients were admitted as emergency. Adequate laboratory investigations, imaging studies, UGI scopy done as a routine for all cases.

OBSERVATION AND RESULTS

In this study following are the observation on analysis.

Table 1

SEX INCIDENCE

Sex	No. of Patients	Percentage (%)
Male	69	69%
Female	31	31%

Among 100 cases admitted in our unit 69% were male and 31% were female.

Table 2

AGE INCIDENCE

Age	No. of Patients	Percentage (%)
20-30	21	21%
30-40	34	34%
40-50	18	18%
50-60	12	12%
60-70	10	10%
70-80	5	5%

On analyzing the age group the age group between 30-40 has higher incidence, incidence decreases with age above 70 years.

Table 3
DISEASE WISE INCIDENCE

Sl.No.	Diseases	No. of Cases
1)	Peptic Ulcer Disease	32
	Duodenal	23
	Gastric	9
2)	Varices	19
3)	Erosive Diseases	43
	Gastric	37
	Oesophageal	5
	Duodenal	1
4)	Drug Induced gastritis	3
5)	Mallory Weiss	1
6)	Malignancy	2

On analyzing the causes it is found that erosive disease is more common with 43% of patients and of these gastric erosion is more common.

Table 4
DISEASE WISE SEX INCIDENCE

Sl.No.	Diseases	No. of Patients	Male	Female
1)	Peptic Ulcer Diseases	33	23	10
	Duodenal Ulcer	23	16	7
	Gastric Ulcer	9	5	4
2)	Varices	19	15	4
3)	Erosive disease	43	31	12
	Gastric	37	27	10
	Oesophageal	5	3	2
	Duodenal	1	1	0

On analyzing this it is found that erosive disease, peptic ulcer disease are more common in male than female.

Table 5

MALIGNANCY

Sex	No. of Cases
Male	1
Female	1

Both patients were managed conservatively and taken up for elective surgery.

Table 6
MASSIVE BLEEDING

Defined as bleeding which needs 3 units or more for resuscitation.

Sl.No.	No. of Transfusion	No. of Cases
1)	1 Unit	43
2)	2 Units	38
3)	3 or more units	19

Most of patients who had massive bleeding were with oesophageal varices and one patient was with duodenal ulcer and one with Mallory weiss syndrome.

Table 7
TREATMENT

Sl.No.	Treatment	No. of Cases
1)	Conservative	82
2)	Endoscopic sclerotherapy	15
3)	Surgery	3

Most of the 83% of cases were treated conservatively by medical management.

Table 8
DISEASE WISE TREATMENT

	Medical	Surgical
Erosive Disease		
Gastric	37	-
Esophageal	5	-
Duodenal	1	-
Peptic Ulcer disease		
Duodenal	22	1
Gastric	9	-
Oesophageal varices	12	2
Drug Induced Gastritis	3	-

In patients with erosive disease all patients (43) were managed conservatively by medical treatment.

Peptic ulcer disease patients all gastric ulcer patients were managed conservatively and out of 23 duodenal ulcer patients except 1 all were managed medically.

Out of 17 esophageal varices patients, 12 patients undergone endoscopic sclerotherapy, 2 patients managed by surgical devascularisation.

All patients with drug induced gastritis managed medically.

Table 9

MORTALITY

Sl.No.	Diseases	No. of Cases
1)	Duodenal Ulcer	1
2)	Varices	3

There were 4 deaths.

One case of bleeding duodenal ulcer expired following surgery.

Three cases of oesophageal varices died. All these patients brought in a state of shock. We have revived and further they collapsed following further episodes of bleeding.

DISCUSSION

Incidence of upper gastro intestinal bleeding is 220 per 100000 cases. Total number of cases admitted during the study period in our hospital is 1,90,000 out of these UGI bleeding was 420 cases and out of these 100 cases were selected for my study which were admitted in our unit at Stanley Medical College.

ETIOLOGY

Among various etiologies enumerated alcoholism and smoking induced and drug induced gastric erosions, peptic ulcer disease and portal hypertension are most commonly noted.

EROSIVE DISEASE

This is the most common cause of upper GI bleeding in this study.

- Gastric erosion more common than duodenal and esophageal erosion.
- Majority of them have significant risk factors like smoking, drug intake, alchoholism, stress.
- All had mild to moderate amount of hemetemesis.
- All were treated medically with no mortality.

PEPTIC ULCER DISEASE

Majority of surgical textbooks and journal indicated that peptic ulcer disease is most common cause of UGI bleeding. In my study it occupies 2nd place. As in the books, DU is more common than GU. Most duodenal ulcers were treated medically.

Majority gastric ulcer occur in incisura angularis and treated medically. Emergency surgery for bleeding gastric ulcer is nil.

OESOPHAGEAL VARICES WITH PORTAL HYPERTENSION

This is third common cause, but many cases with available investigations the cause was not detected.

Cirrhosis with portal hypertension is mainly admitted in medical wards and managed conservatively. Endoscopic management predominate than surgical intervention in cirrhosis with bleeding.

CLINICAL PRESENTATION

Severe bleeding and shock at initial presentation is more common in portal hypertension. In other disease incidence of severe bleeding is less than that of portal hypertension.

Presence of co morbid medical disease such as diabetes mellitus, hypertension, Ischaemic heart disease, myocardial infarction, respiratory disease are equal in all causes of UGI bleeding. They produce fatal

outcome particularly in portal hypertension probably due to massive bleeding.

INVESTIGATIONS

- Routine investigations along with platelet count done in all patients.
- Liver function tests abnormal with varying severity.
- Most patient's comes under child pugh's criteria "B".
- They are the best candidates for surgical interventions.

UPPER GI ENDOSCOPY

- Done in all cases
- Majority show gastric erosion with gastritis.
- Next common is duodenal ulcer in first part and gastric ulcer in incisura angularis.
- Of oesophageal varices , grade II varices more common

MALIGNANCY

- Incidence is 2%
- Not uncommon causes of bleeding
- 1 patient male and one female.

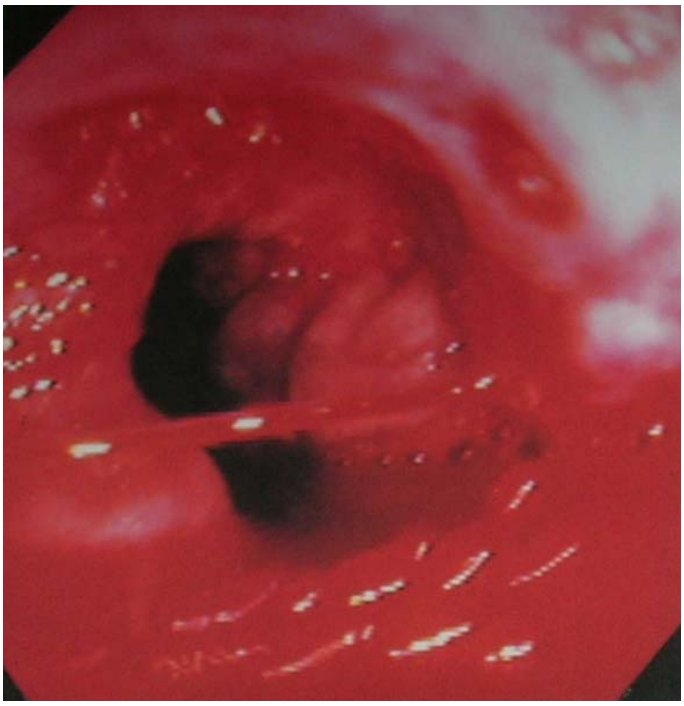
Over all mortality 4% and these were patients with portal hypertension and with bleeding duodenal ulcer. All patients had comorbid medical diseases such as myocardial infarction, hypertension, diabetes.

CONCLUSION

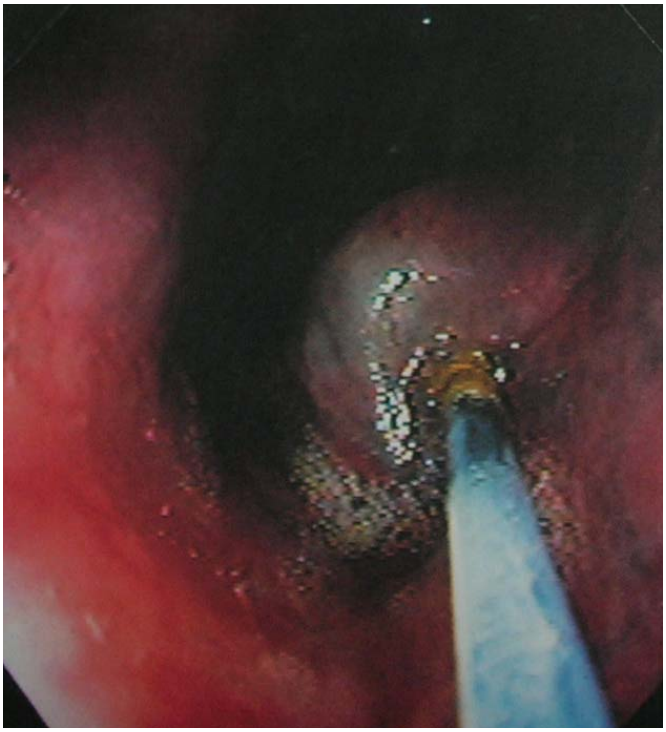
- Most patients were males (69%)
- Erosive disease most common (43%) cause of UGI bleeding in our hospital.
- Of erosive disease gastric erosions with gastritis is more common.
- All erosive disease patients were treated medically
- Second common cause is peptic ulcer disease (33%).
- In peptic ulcer disease, duodenal ulcer (23%) is more common than gastric ulcer (10%).
- Most duodenal ulcer patients were treated conservatively except one with bleeding ulcer treated surgically by suturing ligation of bleeding artery.
- All gastric ulcer patients were managed conservatively.
- Third common cause is oesophageal varices (19%).
- Most patients were treated by endoscopic sclerotherapy(12%)

- Patients with oesophageal varices presented with major bleeding requiring more than 3 units.
- Malignancy not uncommon cause for UGI bleeding.
- Mortality nil in erosive disease and peptic ulcer disease.
- Death more common in oesophageal varices with portal hypertension, mainly due to massive bleeding and co-morbid diseases.

BLEEDING OESOPHAGEAL VARICES

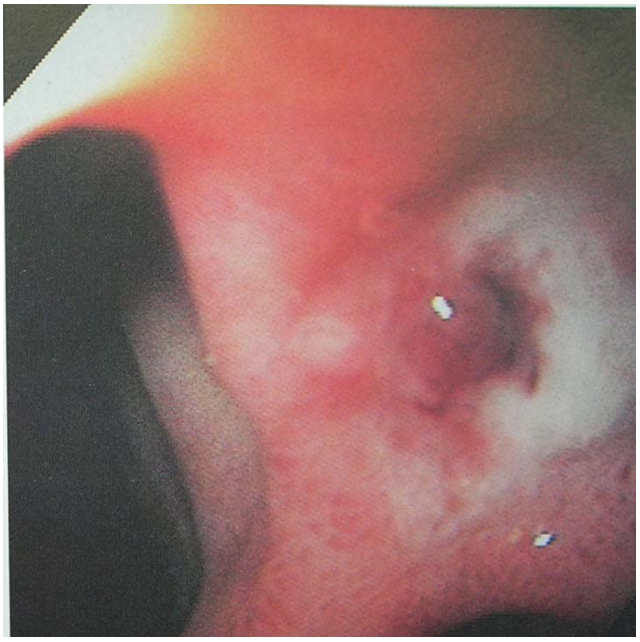


ENDOSCOPIC SCLEROTHERAPY FOR VARICES

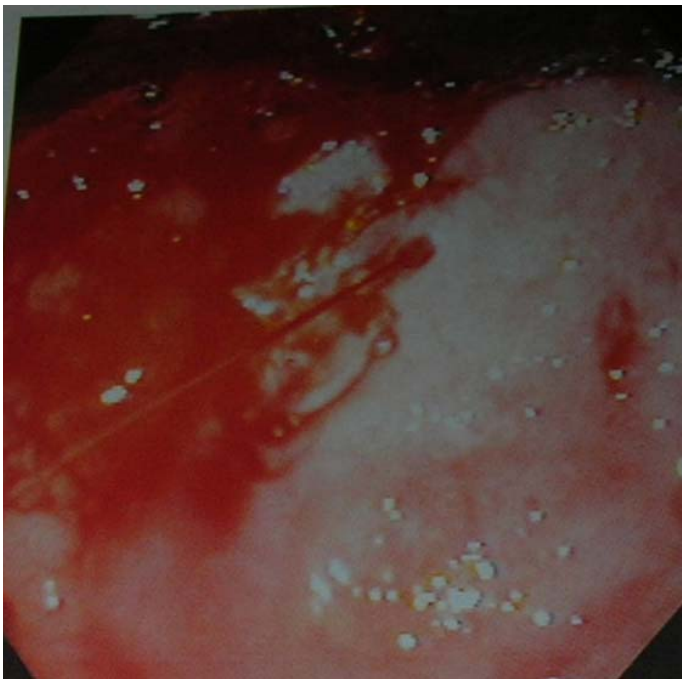


DUODENAL ULCER

DUODENAL ULCER WITH VISIBLE VESSEL



BLEEDING GASTRIC ULCER

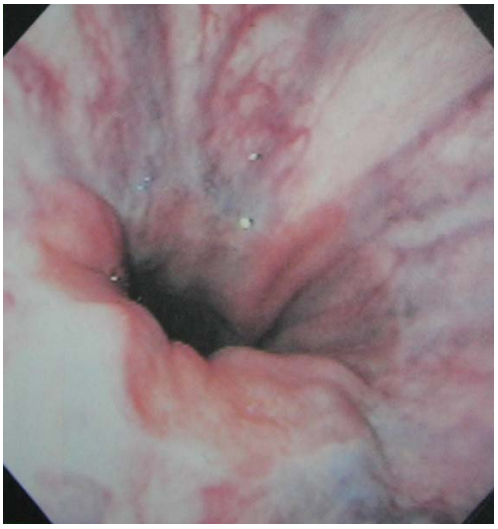


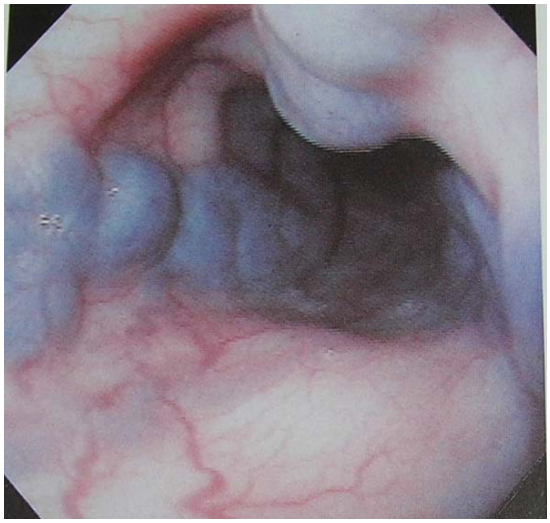
HEAT PROBE APPLICATION FOR BLEEDING ULCER

OESOPHAGEAL VARICES

OESOPHAGEAL VARICES

GRADE 2

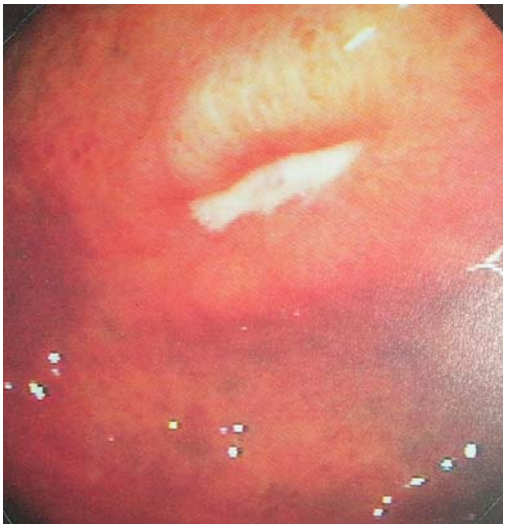


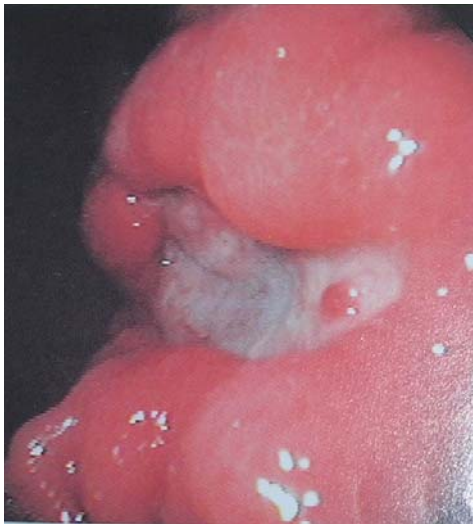


OESOPHAGEAL VARICES
GRADE 3

NSAID INDUCED

BENIGN GASTRIC ULCER





MALIGNANT GASTRIC ULCER WITH VISIBLE VESSEL